Chapter 10 - Treatment Protocols for Low Back Pain Syndrome

Treatment protocols must relate to whether the low back pain is acute or chronic. Prevention of recurrence falls into the category of treatment.

\textit{Pain is the predominant symptom bringing the patient to the health provider.} Pain remains the focus of most care, and chronic pain the most prevalent factor of persistent impairment and disability. Pain is a warning signal that helps protect the body from tissue damage. Sherrington defined pain as a psychological adjunct to a protective reflex, the purpose of which is to cause the affected tissues to be withdrawn from the potentially noxious (and injurious) stimuli. Pain, however, also has a psychosocial aspect that will receive significant attention!

Sensation of pain originates from the activation of nociceptive primary afferents by intense thermal mechanical or chemical stimuli. These nociceptive sites are small, free nerve endings in the numerous tissues of the body.

The acceptance of pain as being exclusively a peripheral manifestation at its receptor sites as a result of tissue damage going directly to the brain is no longer tenable (Fig. 10-1). Originally pain was considered to be dependent solely on the intensity of the noxious stimulus, but this has been refuted.

The concept that specific nerve endings in the involved tissues were also specific in the sensation translated has also been refuted. Concept postulated that specific nerve endings produced specific pain sensations. It is now known that there are specific nerve types that transmit sensation that will ultimately be interpreted as pain.

Normal forces on skeletal tissues, both external and internal, cause tissue deformation (see Fig. 4-8), which is reversible when these forces are removed. When excessive, these forces cause irreversible tissue damage, which ends up liberating nociceptors (see Fig. 4-6).

There are specific nerve types that transmit sensation that will ultimately be interpreted as pain. These nociceptors, pain-producing substances termed algogens, are released at the site of tissue injury.

Leukotrienes are one of the liberated algogens as are phospholipids that break down to arachidonic acid and prostaglandin E. Local tissue trauma breaks down blood platelets that release serotonin, which acts as a vasoconstrictor, causing local edema. Other algogens are histamine, substance P, somakinin, vasoactive polypeptides, and cholecystokinin, to mention a few. Many are vasodilators or vasoconstrictors as well as irritants.

In the low back, the edema causes compartment compression, as the erector spinae muscles are contained within partially inflexible fascial sheaths (Fig. 10-2).

The enclosed nerves contained within their individual sheaths are compressed, causing intra-arachnoid pressure, which can cause temporary transmission inhibition and when prolonged can cause intraneural fibrosis.

Transmission of nociceptive impulses is now well accepted as being via unmyelinated C fibers and lightly myelinated A alpha fibers (Fig. 10-3) which ascend to the dorsal root ganglia (Fig. 10-3) on their way to dorsal gray columns, with 80% of afferent nerves transmitting impulses via unmyelinated nerves as pain sensations. Unmyelinated nerves transmit impulses slowly. The large-diameter, myelinated, faster-transmitting fibers transmit stimuli such as touch, temperature, and proprioception.
The dorsal horn is divided into laminae (rexed layers) (fig. 10-4) into which nociceptive fibers synapse in laminae I to IV to fibers that ultimately traverse the cord to ascend the lateral spinal thalamic tracts (fig. 10-5) to the thalamic region (fig. 10-6).

The A delta fibers transmit faster and carry sharp pain.

The C fibers are slower and carry dull, longer-lasting pain.

There are intrinsic factors in the central nervous system that modulate pain. Endogenous opioid substances are now considered to be synthesized by and within the nerve cells. These substances mimicking narcotic and analgesics are termed endorphins (enkephalins).

The question was raised, "Why does the brain need this pain-modulation system as well as at the dorsal root ganglion, spinal cord (dorsal horn), midbrain, hypothalamus, periaqueductal gray area, and at the rostral medulla.

The International Association for the Study of Pain (IASP) has characterized neural pain mechanisms as along five axes:
- Site of pain
- Physiologic system
- Temporal pattern and recurrence
- Intensity and duration
- Etiology

Theories about the physiologic system postulate that all messages are transmitted within the nervous system as electrical impulses and act across numerous synapses (fig. 10-7). These messages are termed neurotransmitters.

Besides transmission through a neural network (fig. 10-8) it is also postulated that electrochemical signals are transmitted via extracellular medium (fig. 10-9).

Stress has been invoked as objectively and subjectively influencing pain perception.

There are two neuroendocrine systems involved in this mechanism: the hypothalamo-pituitary-adrenocortical and the hypothalamo-sympatho-adrenocortical systems.

The concepts of pools within the nervous systems has been advanced (fig. 10-11). These pools also indicate the sites of action on the treatment of pain (fig. 10-12)!!!

[Elaborating on Fig. 10-12]:
- Anti-inflammatory agents (steroids and NSAIDS) act at the tissue site of injury.
- TENS acts at the peripheral area from the tissue site and the dorsal root ganglion (DBG) prior to entry at the cord level (SG).
- A sympathetic nerve block (block) interferes with sympathetic impulses originating at the sympathetic ganglia (GGL).
- Epidural steroids act at the cord level of ascending lateral spinothalamic tracts (LSTT).
- Tricyclic medication acts on the thalamic, hypothalamic (limbic system) level whereas
- Psychotherapy intervenes at the level of the cerebral cortex.
The reflex muscular spasm that accompanies local pain also can become a site of nociception (fig. 10-13) that aggravates the pain and leads to disability (fig. 10-14).

The final pathway is the brain, where the pain is interpreted.

To date there is no brain measure that correlates neurophysiological activity to clinical pain, but there is promise.

Chronic pain was originally considered to be pain that lasted longer than 3 months, but chronic pain is now considered to exist in significantly shorter periods of time.

Proper treatment of acute pain can prevent chronic pain before it becomes deeply engrammed in the central nervous system!

Acute pain must be addressed in all its aspects, otherwise chronicity and disability will prevail (fig. 10-15).
All interventions to minimize the chemical, mechanical, and electrical effects of the algogens on the neurophysiological conduction of nociception must be applied.

This must be done with the appreciation of the psychosocial implications of the pain from the very onset. Treating the symptoms on the basis of a purely mechanical chemical basis from impaired neuromusculoskeletal mechanism is fraught with ultimate failure.

The patient must be treated, not merely the symptoms, the clinical findings, or the diagnostic tests. The acute symptoms must be addressed, but always with the need to instruct the patient of the meaning of the symptoms, the significance of the resultant disability, and the role to be played by him or her as the procedure ensues.

The noxious agents that accumulate at the peripheral trauma site, comprising histamine, kinins, neuropeptides, and numerous other algogens, have a vasomotor effect (either vasodilation or vasoconstriction).

These nociceptive agents lower the threshold of the receptors of the A delta and C afferent fibers of the peripheral nervous system, sending impulses cephalad. Intervention or elimination of these algogens at the trauma tissue site is the objective of the application of most modalities.

The local area becomes hypersensitive, creating pain known as primary hyperalgesia. A secondary hyperalgesia occurs in the surrounding tissues from antidromic activation of the C primary afferents, which release substance P in the region.

Electrical stimulation of the primary afferents has been shown to release substance P at their receptor ends

Reactive local skeletal muscle spasm occurs, initiating a pain-spasm-pain cycle

The chemical and mechanical substances produced at the peripheral tissue site following injury are mechanisms of pain production that must be addressed in the treatment of acute pain
PHYSICAL MODALITIES

CRYOTHERAPY
Cryotherapy, the application of cold in the treatment of acute treatment of pain, has been an accepted modality for centuries. Cold lowers the temperature of the skin and underlying tissues essentially by removing heat from these tissues.

Theories behind its local application as a treatment modality for a traumatized area are that:

1. it decreases or inhibits bleeding
2. it decreases local tissue metabolism, which produces algogens
3. it neutralizes the local histamine liberated by trauma
4. it decreases local muscle spasm by decreasing the sensitivity of the muscle spindle system
5. it elevates the threshold of pain-transmitting nerves

A local tissue reaction to trauma is the formation of local edema. Edema occurs because of change of hydrodynamics as the vasoconstriction is followed by reflex vasodilation.

The afflicted vessels, the arterioles, the capillaries, the venules, and the lymphatics become distended. The endothelial cells separate, creating gaps between the cells and allowing greater filtration of the serum with its contained constituents to enter the perivascular tissues.

This edematous fluid at first is merely a transudate containing water and dissolved electrolytes, with a specific gravity of 1.012, maintaining osmotic balance. As permeability increases, the transudate becomes an exudate, containing cells and proteins with a specific gravity more than 1.012.

Both the transudate and the exudate cause a mechanical impedance to further blood flow, resulting in ischemia.

The protein contained in the exudate gradually causes chemical "thickening" of the fluid, which impairs physiological movement between fascial planes.

Ice or cold applied to the inflamed tissues intervenes in this transudate-exudate cycle by decreasing fluid transudate and decreasing the metabolic rate.

Cold also decreases tissue sensitivity, which permits active and passive exercises that mechanically eliminate the exudate and transudate from tissues!

Nerves differ in their reaction to cold, depending on their degree of myelination. The unmyelinated, small-diameter fibers are less responsive to cold than are A fibers, with the large motor fibers (alpha) the least affected.

Exercise performed after the application of cold generates more muscular tension. The combined effect of ice, decreases pain, alters the hydrodynamics, and permits greater strength of muscle contraction, decreasing the accumulation of edema and removing the accumulation of the nociceptive metabolites.

THERAPEUTIC HEAT
The sequelae of the effect of heat on tissues depends on the extent of temperature rise, the rate of application of heat energy and the volume of tissue exposed to the heat application.
Elevation between 40°C (104°F) and 50°C (122°F) increases blood flow, which is the therapeutic objective.

The rate of temperature rise influences its efficacy. The rate of temperature rise influences its efficacy. A slow rise of tissue temperature may defeat the objective of heat application, as it brings cooler blood into the inflamed tissue site. Too rapid a temperature rise may also be deleterious, as the heat generated in the local tissues may stimulate pain receptors with adverse effect.

The effects of heat can be stated to be an alteration in metabolic activity, hemodynamic function, neural response, skeletal muscle activity, and modification of collagen tissue. All these effects can be directly or indirectly related to the management of pain resulting from tissue trauma. The neural response more directly intervenes in transmission but the other effects of heat relate to the tissue dysfunction, which also enhances pain. The neural effect of how heat provides analgesia and reduces muscle spasm, both involved in pain production, is not fully understood. The latter, reduction of muscle spasm, is conceivably induced via the spindle system.

Surface heating agents do not elevate muscle temperature needed to alter II or I b afferent nerve activity, whereas skin temperature heating has decreased gamma efferent activity, which may relate to diminished muscle spasm related to pain reduction.

Skin temperature heating has decreased gamma efferent activity.

Metabolic rate increases two to threefold with every 10 °C rise. Increasing tissue temperature above 50 °C (122°F) burns the tissues!

Moist heat transmitted via hot moist packs has many advocates. Local hot paraffin wax is effective in treating extremities, as are hydrotherapy, ultrasound, diathermy pulsed electromagnetic fields, and laser.

The choice of source of heat will depend on the training and experience of the physician, or empirically.

SOFT TISSUE MODALITIES FOR ELONGATION

Connective tissue, which is so often impaired after injury or disease, is benefited by heat application.

The viscoelastic property of connective tissue that permits elongation from physical stretch is known as plastic deformation.

The need to regain tissue flexibility in treating pain is apparent in that sensory nerves are enclosed within the soft tissues, which often have become impaired after injury or prolonged tension from anxiety, anger, and emotional tension.

To regain the physiological elongation of damaged tissues to their normal length requires ensuring the appropriate temperature elevation as regards intensity, site, and duration.

Consideration must also be given as to the extent of physical stretch regarding its intensity, duration, and
velocity, from (1) constant load to overcome impaired elasticity, to (2) rapid stretch followed by holding the gained elongation, to (3) a slow progressive stretch.

**ANALGESIC NERVE BLOCK IN TREATING PAIN**
This concept is valid to acute and recurrent pain for its diagnostic localization, treatment, and even prognosis.

Diagnostic blocks are valuable in conforming structural abnormality.

*The purpose of analgesic nerve blocks is to interrupt the transmission of nociceptive impulses of the afferents to the cord from the damaged tissues.*

Diagnostically the afflicted organ or tissue is identified by interruption of the somatic nerve to and from that tissue.

Analgesic blocks are effective in relieving muscles spasm. Interruption of the afferent impulses in the treatment of acute pain allows a more normal healing process or, at least, allows comfort to that patient during the healing process.

The analgesic blocks are administered into the tender muscle and into the ligaments of the apophysial, sacroiliac, and sacrococcygeal joints (fig. 10-16), which do not interrupt the nerve supply to the affected tissues.

Of the neural blocks, the caudal and peridural blocks are effective.

It is well documented that the analgesic value that occurs during the presence of the injected drug continues after the chemical duration of the drug.

There is benefit from insertion of a needle without the use of medication! The needle used here may be a placebo or effective as to its neurophysiological aspect. The pain relief obtained must be evaluated carefully to avoid ascertaining that the nerve blockade of the pain was objective and conclusive. "The placebo effect . . . is most powerful when a trusted physician enthusiastically offers a patient a new therapy."

Discogram, injection into the nucleus of a disk, has been advocated as a diagnostic test.

Surgery for lumbar spine pain or low-back pain with radiating leg pain has a notoriously unsuccessful track record.

Injections are limited to diagnostic procedures, facet etiologies, discograms, and epidural analgesia.

The technique of epidural injection is well documented (fig. 10-17).

Epidural corticosteroid injection has proved to be a relatively effective treatment for low back pain and sciatica.

**EXERCISE IN TREATMENT OF LOW BACK PAIN**
For many years exercise has been the major conservative modality in the treatment of low back pain being advocated for acute pain, for the prevention of recurrence, and even for the management of chronic low back pain.

There persists a controversy as to which exercise is appropriate in treating the various back pain syndromes. There are advocates of flexion exercises, extension exercises, aerobic exercises, stretching regimens, diminution of tension, and exercise as an aspect of normal low back function. The controversy is augmented in respect to active versus passive therapy.

The literature on exercises in relationship to low back pain has had the following objectives:
- To decrease the duration of the impairment and thus disability
- To strengthen and increase endurance
- To reduce mechanical stress
- To correct posture
- To bring about general restoration
- To reduce pain

Increasing strength and endurance of back muscles have become the cornerstones of treatment for low back pain. The duration of back pain appears to correlate with the resultant muscle strength and endurance. There was an insignificant difference in strength between normals and patients with symptoms for less than a month.

Reduction of mechanical stress (ergonomics) with exercise also remains controversial. "Body mechanics" involve eccentric muscle contraction—elongation and deceleration (see Chapter 1). Strengthening exercise directed at the abdominal (flexors) allegedly decreases the load upon the disks, remains unconfirmed.

A concept of the value of the oblique abdominal muscles effect of the oblique muscles upon the fascia (Fig. 10-18). This concept implies that the value of the exercise is not during the lifting but as a strengthener of the tissues used during a lifting episode.

Poor posture, when it is considered a cause of low back pain, supposedly is improved by exercise, but this remains unconfirmed.

During acute pain exercise generally is contraindicated except for maintaining self administered passive range of motion (ROM) of all extremities and the trunk. Subacute pain, however, is less intense therefore therapeutic exercise is highly desirable and realistic for restoration of function to the affected area.

In the treatment of chronic pain, exercise is directed to the effects of decreased activity, leading to atrophy, weakness, contracted joints, and so on, with pain only indirectly addressed.

There is no doubt that muscular weakness, prevalent in many musculoskeletal pain syndromes as well as fatigue and debility (physical weakness) that occur in depression from chronic pain, can be altered, in part, by exercise. In that respect, exercise to regain strength and endurance as well as flexibility and mobility is a powerful adjunct in the treatment of pain.

It has been implied that exercise increases the level of endorphins (endogenous opioid peptides), which are accepted neurotransmitter with a morphine-like action. This modulation of pain perception and analgesia has been associated with the analgesia of electrical brain stimulation and acupuncture.
Exercise results in elevation of adrenocorticotropic hormone, Cortisol, and catecholamines, which are the precursor of beta-endorphins. Many athletes have had a decreased pain perception that has allowed them to make maximum effort in spite of pain and even claimed to reach with a morphine-like action. This modulation of pain perception and an emotional high after extreme physical activity

Muscular deficiency could be a causal factor in low back pain and that muscular fatigue could also be significant.

Strengthening exercise directed at the abdominal allegedly decrease the load upon the disks.

A concept of the value of the oblique abdominal muscles is the effect of the oblique muscles upon the fascia (fig. 10-18).

Exercise must be strenuous to be analgesic. Exercise has a well-documented place in the physical treatment of most neuromusculoskeletal painful disabling conditions, but its precise role in the treatment of acute low back pain remains unclear.

We must tell our patients of the good prognosis following an attack of acute low back pain, we must instruct them in what to do, how to move and of the beneficial effects of mobilization towards work." Exercises in this respect are a mandatory aspect of the treatment protocol.

Gradated, supervised, specifically prescribed exercises also allay the fear of recurrent from any movement or activity. The fear of recurrence from any activity is prevalent and must be addressed in treating the patient with acute low back pain. Exercise addresses this if there is concurrent reassurance and explanation by the physician in meaningful words and examples. The possible discomfort of exercise must be explained and justified.

Walking, as an exercise prescription, is probably the simplest, least stressful, and most beneficial therapeutic exercise.

**Specific Exercise Protocols**  
Muscles and their fascia need repeated passive and active elongation to maintain their physiological length.  
Tendons and ligaments are also in that category, as are the capsules of synovial joints. The collagen fibers are the basis of maintained elongation, as are fibrous and elastin tissues.

Even the annular fibers of the intervertebral disk annulus, consisting principally of type I collagen fibers with an increasing percentage of type II fibers, are in need of physiological elongation for their nutrition.

In a tight low back, the trunk does not fully flex either in forward flexion, lateral flexion, trunk rotation, or extension.

Limited flexibility may be the result of pain or may be the sequela of restricted physiological elongation of the soft tissue from disuse.

Restoration of flexibility after an acute episode is usually attempted, along with a strengthening program. Proper spinal alignment demands total flexibility to the physiological limits of the component tissues.
Exercise often benefits from the application of heat or ice, depending on the indications.

Flexibility exercises aim at improving disk nutrition and growth of the collagen fibers within. Self-directed stretching and mobilization programs correct the fascial, muscular, ligamentous, and capsular shortening. As most of the motion (75%) of the lumbosacral spine occurs at L5-S1, if that segment is restricted it will cause excessive motion at other segments [hypomobility causes hypermobility!], in this case at L4-5 segments, with degenerative changes of these segments enhanced from increased shear.

In total spinal flexibility the pelvis must be addressed to ensure a correct lumbar pelvic rhythm. Pelvic flexibility includes the glutei, hamstrings, and the gastroc-soleus, which must be elongated without increasing stress on the lumbosacral spine during the exercise. This indicates protective hamstring stretching (fig. 10-19).

Flexibility and strengthening exercises also address restoration of proprioception. Proprioception is the transmission of joint position information in complex activities.

*Physical response of dense connective tissue to therapeutic stress is the basis of stretch exercises.*

The immediate response to stretch may be a transient increase in tissue length, which indicates why exercise must be done repeatedly and consistently.

The responses of tissue to elongation activities are itemized in the following:

1. Collagen fibers that are elongated 1% to 1.5% for less than 1 hour show no permanent deformation
2. Elongation of 1.5% to 2% maintained for more than 1 hour will result in permanent elongation because that degree of stretch results in a melting of the tropocollagen bonds. The gain may be lost if the elongation is less than 1 hour or if the tissues are allowed to decrease their elongation.
3. Elongation of 2% may allow return to prestretch length if not followed by sustained or intermittent stretch during the subsequent 24 hours.
4. Elongation of 3% to 8% may cause a loss of continuity of the collagen and result in damaged or inflamed tissue.
5. Permanent stretching or excessive elongation tears the structure of the collagen fiber or disrupts the intermolecular bonds between the tropocollagen units, causing permanent damage to the collagen fibers.

*Flexibility exercises should be done gently, repeatedly, eliciting minimal and self-limited discomfort with no effort to exceed full range of motion as determined by the patient rather than by the therapist using physiological standards.*

*In summary, immobilization causes a significant loss of dense connective tissue strength, predisposing it to damage on ultimate elongation. Allowing tissue shortening for any length of time in a soft tissue injury is thus to be avoided. Excessive and prolonged stretching after injury and its immobilization must be judiciously applied. The biomechanical properties of collagen must be kept in mind when applying stretch exercises. Passive (done to) stretching is far more dangerous than active (done by) stretching.*

Self-applied stretching exercises are the best prescription for the patient with low back pain.
Walking remains the best form of exercise (Figs. 10-20 and 10-21) because it addresses every aspect of body physiology. All tissues are involved, ensuring their physiological elongation, including the annular fibers of the intervertebral disks (Fig. 10-22). These benefits are also enhanced by the cardiovascular pulmonary and psychological benefits of rapid walking.

Active exercise to regain or maintain flexibility can be done daily without the use of mechanical equipment (figs. 10-23, 10-24, and 10-25). These exercises stretch the lateral ligaments (fig. 10-26) and the fascia of the erector spinae muscles.

Rotational exercises are also mandatory, providing they are within the elongation limits of the annular disk fibers. Because the collagen fibers of the annulus are oriented in different directions, only half of the fibers resist clockwise rotation.

Disks are stiffer in torsion than in bending. During axial rotation, the increased stress in the collagen fibers raises the hydrostatic pressure in the nucleus pulposus, but this pressure is significantly less than occurs in bending. When there is a combination of compression and torsion, the disks completely recovers if rotation does not exceed 9 degrees.

Normal disks completely fail when rotation exceeds 10 to 26 degrees. At failure, the outer lamellae of the annulus separate from each other and tear away from their endplate attachments. Torsion does not damage the nucleus or inner annular fibers and does not cause radial fissures, so it does not cause disk prolapse.

Gentle rotational exercises (fig.10-27) are physiologically beneficial.

As a combination of flexion and rotation occurs repeatedly in most activities of daily living, exercises to maintain this flexibility are of value (fig.10-28). These exercises also enhance the proprioceptive aspects of the motion.

Limited elongation of the hamstring muscles imposes excessive stress upon the lumbosacral spine in flexion activities. Exercises to elongate these muscles and their fascia must be undertaken repeatedly in a slow manner. These exercises must take into consideration the effect on the low back and thus be what can be termed unilateral “protective hamstring stretch exercises” (fig. 10-25).

The heel cords must also be frequently stretched, as they impose limited total flexion as well as limited hamstring elongation (Fig. 10-29).

The hip flexors have also been impugned in causation of low back pain and should be evaluated and, if found limited, be elongated to their physiological limits (Fig. 10-30).

**Strengthening Exercises**

Strengthening exercises of the muscles of the trunk have been advocated by all therapists treating low back pain syndromes.

The traditional abdominal flexor strengthening exercises have been the sit-up, the sit-back, and bilateral straight leg raises (figs. 10-31, 10-32, 10-33, and 10-34).

The concept of the "air bag" unloading the spine was postulated as a basis for unloading the spine but was refuted as the needed intra-abdominal pressure exceeded the pressure within the abdominal blood vessels. A more acceptable concept is the effect of the oblique abdominal muscles on the thoracolumbar fascia, creating a compartment of the erector spinae muscles. The action on the fascia from widens the
fascia and creates a compartment that unloads the spine. (Fig.10-23). This concept implies the need to strengthen the oblique abdominal muscles as well as the sagittal flexors.

**Inappropriate Abdominal Flexor Exercises**
The traditional abdominal flexor strengthening exercises have been the sit-up, the sit-back, and bilateral straight leg raising. The last mentioned is appropriate only in people who have adequate abdominal muscles, as this exercise (Fig. 10-31) is often done with simultaneous hyperextension of the lumbar lordosis, which may cause discomfort (Figs. 10-32 and 10-33).

The ideal sit-up exercise to strengthen the abdominal muscles is performed with the legs flexed at the hips and knees. The exercise is done in stages. Initially the head is flexed, which involves the neck flexors. This initiates synchronous total flexors to contract. The next phase is to elevate the thorax and gradually progress to a total sit-up (Fig. 10-34). Essentially a total sit-up is not necessary. Merely to elevate the thorax from the floor contracts the abdominals to a full contraction. The later sit-up stages become redundant.

The sit-back exercise begins with the person fully flexed both at the legs and the trunk and involves a gradual descent backward done in stages and degrees (Fig. 10-35). At any stage the body can be held, causing an isometric contraction, which adds endurance to strengthening. This exercise is ideal for the poorly conditioned, as it is easier and also addresses eccentric muscular activity.

Stress should be placed on strengthening the oblique abdominal muscles.

Exercises with trunk rotation strengthen the oblique abdominal muscles (figs. 10-36, 10-37, and 10-38).

The purpose of strengthening abdominal muscles as well as unloading the spine is to decrease the lordosis (fig. 10-39) and improve the posture.

Based on the conclusion that annular disk tears occur from combined rotational, flexion, shear, and compressive forces, exercises implementing these actions must be done slowly, gradually, and progressively and always under careful control.

In the treatment of alleged lumbar disk herniation as the cause of low back or leg pain, flexion exercises have been widely advocated.

In the treatment of alleged lumbar disk herniation as the cause of low back or leg pain, flexion exercises have been widely advocated. The concept of flexion exercises as opposed to extension exercises was challenged by MacKenzie, migrate anteriorly and posteriorly within the intervertebral disk by various spinal movements. Of special interest was that the posteriorly protruding nucleus against the posterior annular fiber protecting the posterior longitudinal ligament and the emerging nerve roots could be caused to migrate anteriorly and thus away from nociceptive tissues (Fig. 10-40). Treatment of a disk herniation was thus postulated to benefit from extension position and exercise (Fig. 10-41).

The value of the pelvic tilting exercise, admittedly a flexion exercise, is not diminished by the MacKenzie concept as it is implemented after anterior migration of the nucleus but extension has been considered to have occurred.

All the preceding exercises are useless if the body is inappropriately and incorrectly used. The exercises must be properly done. Properly implies the procedure being physiological and under the conscious or subconscious control of the person.
Back schools is defined as education and training using the principle of body mechanics and back health care for prevention and control of back pain in the most efficient manner. *Because many injuries to the low back come from a lifting activity, proper bending and lifting are major subjects taught in the back school* (figs. 10-47 to 10-50).

Ergonomics have also held prominence in the concept of proper body mechanics (figs. 10-42 to 10-46).

Improper bending and lifting are also discussed (figs. 10-51 and 10-52).

**CONSERVATIVE CARE OF THE HERNIATED LUMBAR DISK**

Surgery for lumbar spine pain or low back pain with radiating leg pain has a notoriously unsuccessful track record. *Studies have shown that time alone may cure herniated disks for which surgery has been indicated.*

*Other studies have shown that aggressive conservative care (aside from time alone) returns as many as 92% of patients with herniated discs to normal activities within a few months.*

Herniated disk nucleus without extrusion could be physically returned to a more anterior position (figs. 10-46 and 10-47) by a passive hyperextension position.

It can be stated that the occurrence and recurrence of low back pain and the prevention of chronic low back pain can be addressed by many modalities directed to diminishing pain and regaining good body flexibility, strength, and endurance as well as ensuring good body mechanics. The cause of low back pain remains mechanical in most cases, beginning with inappropriate movements, which result in pain and thus leads to disuse, disability, drugs, and depression. The tissue sites of resultant pain have been addressed and the plea remains that the prescribed treatments be meaningful, appropriate, physiological, and adequately evaluated to avoid excessive, prolonged, and non-beneficial outcomes.

**MANIPULATION**

Manipulation varies from gentle stretching to forceful application of manual force. There are many benefits of manipulation.

Manipulation varies from gentle stretching to forceful application of manual force. The latter is *manipulation* and the former is termed *mobilization*.

The benefits from manipulation have been postulated to include:

1. A facet becomes immobilized by an acute synovial reaction, and adherence of the adjacent facet joint surfaces from an inappropriate motion or an abnormal external force. Manipulation separates these surfaces.
2. A meniscus that normally exists within the facet joint becomes entrapped from an unphysiological motion.
3. The redundant facet capsule becomes lodged between the adjacent articular surfaces.
4. The mechanoreceptors of the joint capsule are desensitized by an abrupt unphysiological motion of the joint, preventing further motion.
5. The spindle system of the involved muscles is impaired by an unphysiological motion. Manipulation allegedly reflexively stimulates and reciprocally relaxes the extrafusal muscles.

6. The involved spinal segments become malaligned by an unphysiological movement and are realigned into physiological position by manipulation. This explains the term *adjustment*.
7. Manipulation is a placebo that benefits the patient by a "laying on of hands."
The physiological movements of joints are limited in their range by the elasticity of the capsules, the periarticular ligaments, and the fascial limits of the contiguous muscles. Joints have an active and a passive range (Fig. 10-53).

Manipulation is usually a force applied in the direction of restriction to regain that lost motion.

The concept of manipulation is to regain mobility, but it has also been advocated as improving the stability of a joint by realignment to its physiological position.

Most joints of the spine are incongruous (fig. 10-54), they do not have intrinsic stability.

A comment on the modality of manipulation is that the basis for it is unproved and that it is a passive therapy with no patient assistance.

**TRACTION**

Traction has been used since the time of Hippocrates yet remains unconfirmed as to efficacy.

Traction has enjoyed a range of evaluations, from being a specific modality for the relieve of mechanical low back pain and reduction of intervertebral disk protrusion to being merely a means of keeping the patient off his or her feet.

Passive traction implies a continuous static force being used for a varying period of time, whereas active traction implies varying forces applied via traction or traction coupled with active exercises.

*Physiological studies suggest that the applied traction weight must be 25% of the body weight to overcome the inertia and resistance of the supine body and achieve any distraction of a lumbar functional unit.*

Traction decreases lumbar lordosis. The effects of this on the lumbosacral spine are summarized as:

1. Opening the intervertebral foramina.
2. Separating the zygapophysial joints.
3. Elongating the erector spinae muscles.
4. "Stiffening" the annular fibers of the intervertebral disk, thereby unloading the internal pressure within the nucleus.
5. Decreasing the length of the nerve roots and their dura, thereby decreasing the tension on them.
6. Questionably reducing any nuclear bulging within the disk, thereby diminishing bulging or protrusion

**Methods of Application**

There are numerous methods of application of pelvic traction, with the most common being that to the supine patient (fig. 10-55).

The angulation of the straps determines the direction of pull on the pelvis (fig. 10-56).

Autotraction, meaning a manually exerted force applied through traction equipment (fig. 10-57), is an active traction.
Manual-mechanical traction has been advocated, especially by chiropractors, using a divided table to which the patient is strapped.

Autotraction, meaning self-applied traction by the patient, also can be accomplished by inversion traction, where the feet or legs are immobilized and the inverted patient uses the body weight for the traction force.

Padded boots with a hook, is one type of inversion traction (fig. 10-58).

Exercises can be performed while in the dependent traction position (fig. 10-59).

An elaborate gravity traction is available (fig. 10-60).

Research and clinical documentation do not validate using pelvic traction, but if a home program is initiated that causes the patient to claim benefit, it is valid.

ANCILLARY MODALITIES
TRANSCUTANEOUS ELECTRICAL NERVE STIMULATION
For many years, transcutaneous electrical nerve stimulation (TENS) has been advocated as a treatment for acute and chronic pain, including low back pain.

In the physical treatment of pain, TENS has been well accepted as being effective. Its neurophysiological basis has also been established. Pain mediation through the C unmyelinated and A alpha lightly myelinated fibers has been established. fibers transmit mechanoreceptor impulses and have a lower threshold at their synapse at the dorsal horn. As they transmit at a faster speed than those of the unmyelinated or lighter myelinated, they arrive at the "gate" earlier. The gate concept implies that these impulses block subsequent pain-producing impulses.

[It would serve you well to do a search on the “gate control theory”]

TENS of low frequency and high intensity of less than 10 Hz has been clinically shown to create analgesia.

Questions have been raised as to whether there is an increase in endorphins from TENS and what is the appropriate frequency of the TENS to elevate this opium peptide in the cerebrospinal fluid. It was determined that low-frequency (2 Hz) stimulation caused an elevation of Met-enkephalin-Arg-Phe-Dynorphin: an opioid peptide in the cerebrospinal fluid. Higher frequency (100 Hz) caused a lesser elevation of a different opioid.

Electrical stimulation has been shown to increase levels of of dopamine, epinephrine, and serotonin, which are established algogens. Electrical stimulation has been shown to decrease nerve action potential of A delta fibers, which are the pain mediators. All these factors confirm a physiological basis for effective pain modulation. The manner, site, and type of TENS are vital for effective pain modulation. TENS is most effectively applied proximally in nerve injuries and the precise site of application must be clinically ascertained. The current wavelength must be determined (Fig. 10-61) as well as its form (Fig. 10-62). A recent report advocated greater relief from the use of ultralow frequency TENS (0.66 Hz) in contradiction to the cur- naloxone, which is an indication of a neurochemical basis.
The efficacy of TENS in treating chronic pain has varied from 12% to 60%. Patients with significant depressive illness complicating their pain receive significantly less benefit from the use of TENS, so TENS cannot be considered to effect pain modulation through its psychotherapeutic benefit.

ACUPUNCTURE
The modality of acupuncture treatment is essentially the insertion of small thin solid needles into the skin, immediate subcutaneous, and muscular tissues in regions considered meridians.

The meridians were interconnected within this vital life energy chi, and allegedly a deficiency of chi was considered to cause pain.

The technique is the insertion of the 20-gauge short beveled spinal needle attached to a syringe that is only partially plunged (fig. 10-63).

_The site of insertion appears to be the major basis for success. The sites of trigger points or motor points have been postulated as the optimum sites for acupuncture._

In 1981 the American Medical Association decided that there was insufficient evidence to conclude that acupuncture had no more effect on pain than placebo or sham acupuncture.

Animal studies are themselves inconclusive, because stress is imparted to the animal during the acupuncture experiment and stress itself is analgesic.

Clinical studies of acupuncture are subjective, as most pain studies are expected to be. The mere belief that acupuncture can be effective influences the patient reaction as to the relief of pain from an application. Studies have revealed that acupuncture is no more effective than TENS at the same tissue sites. Cultural background as an influence on the benefit from acupuncture has been studied and found not to be a significant factor.

EPIDURAL ANALGESIA
Epidural analgesia, often with steroids, has been used for many years in treatment of pain and especially in chronic low back pain or low back pain with radicular pain that does not respond to the usual conservative managements.

A narcotic or analgesic produces analgesia by two mechanisms. First, a portion of the drug crosses the dura mater and enters the cerebrospinal fluid, where it penetrates into the dorsal horn of the spinal cord. Second, there is systemic absorption as occurs from intramuscular injection.

The technique is the insertion of the 20-gauge short beveled spinal needle attached to a syringe that is only partially plunged. As the dura is approached through the thick ligamentum flavum and penetrated, the plunger of the syringe is depressed (into the barrel), whereas if the penetration proceeds further into the dural space, the spinal fluid has a positive pressure that ejects the plunger and fills the syringe (Fig. 10-63). Once the epidural space is entered, the injectable fluid is entered. The basis of the epidural route as compared with intradural or peripheral nerve block is illustrated in Figure 10-64. _The injected analgesic and steroid act on the afferent neurological pathways_ (Fig. 10-65), affecting the low back and sciatic radiculopathy.
Nerve root injections have also been used for diagnostic attempts to determine the exact nerve root level in radiculopathy syndromes. Steroids injected into the paraneural area of the nerve root (Fig. 10-67) or into the dorsal root ganglion also have their advocates (Fig. 10-68).

Facet injection of an analgesic or steroid to relieve pain asserted to be emanating from the facets has been discussed in Chapter 6.

Injections into the epidural space and injection of the nerve roots should only be considered when noninvasive modalities have failed and there are organic signs found causing the impairment leading to disability.

**PHARMACOLOGICAL TREATMENT OF LOW BACK PAIN**

The most commonly used drugs in the treatment of low back pain include pure analgesics, NSAIDS, steroids, muscle relaxants, and anti-depressants. Narcotic analgesics are used only when there is severe pain not responding to the lesser analgesic medications.

Analgesics are agents that are considered to relieve pain by acting centrally. A possible mechanism has been postulated by the discovery of opiate receptors in selected area of the central nervous system and of endogenous substances termed enkephalins (endorphins). The receptor sites have been termed mu, delta, and kappa.

Nonsteroidal anti-inflammatory drugs are numerous and must be used with concern because of their potential undesirable side effects, especially gastrointestinal reactions. *Most of these drugs have not proved to exceed the benefit from aspirin.* Muscle relaxants have been nonspecific, as most drugs in this category are also sedative or antianxiety drugs. The drugs used for treating neurological spasticity (such as baclofen) have been used, but the literature varies as to their efficacy.

Carisoprodol is superior to alternative medications.

Sedatives and antianxiety drugs, therefore, have not been proved more effective than other medications and modalities.

Antidepressants have been increasingly used in the treatment of patients with acute as well as chronic low back pain not only for their action against the prevalent depression that accompanies the usual low back pain but also as they have proved to be chemically effective in pain management.

The presence of opiate receptors indicates that the body is capable of manufacturing its own narcotic-like substance.

It is hypothesized that opioids inserted at these sites activate the descending pathways to the midbrain periaqueductal gray matter which inhibits ascension of nociception from the cord level.

Opioids, as well as acting upon the mu, alpha, and kappa receptors, also act upon the postganglionic sympathetic terminals that block release of prostaglandins, which are thought to be involved in sympathetic maintained pain syndromes.

Opiates are markedly underused in treating pain, causing patients to suffer needlessly. This is partly due to the myth of opiate tolerance. The problem has been compounded by restrictive laws regarding opiates, with attendant social, political, and legal problems. This is not to advocate the indiscriminate
use of opioids but to state that brief, carefully moderated use of opioids in patients suffering severe acute low back pain may lead to earlier recovery and resumption of beneficial activity.

The use of opioids in the treatment of chronic low back pain is more complicated.

A dictum must be applied that medications be appropriately administered.

Benefit will be derived from a precise period of time and specific dosage of medication.

Current concepts of treating chronic pain mandate that the initial intent be to decrease or eliminate the dependency of the patient upon pain medications, especially if they are addictive or become less effective from prolonged use. If dependency on drugs is initially avoided, their later elimination in treating chronic pain results.

A series of chemical reactions in nociception occurs within the central nervous system that once established may perpetuate and increase. This indicates the need for early intervention based on a full understanding of the mechanisms of pain production and transmission (fig. 10-69).